Stress and Nutritional Quality of Broilers[1,2]

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ABSTRACT Broiler chicks were reared in environmental chambers. All birds were started under ideal conditions, i.e., 30.6 C with 35% RH. Beginning at Day 36, half of the chicks were maintained at 24 C and 35% RH. The other half were subjected to a cyclic temperature-RH regime that approximated a typical August day in central Mississippi (heat treatment). Half of each of the described groups received implants of osmotic pumps that released adrenocorticotropin (ACTH) at 8 IU/kg BW/d for 7 d. The remaining birds received placebo pumps. The main effects of ACTH and heat treatments were similar. Both treatments caused reductions in BW, carcass weight (CW), carcass protein (CP), and muscle calorie (C) content. ACTH, but not heat, reduced carcass moisture (M). Carcass fat and ash, however, were not affected. Most changes were not reversed after 1 wk of recovery. Although visible signs of pale, soft, exudative muscle (PSE) were present, "white" areas of muscle were absent. The decreased meat yield and detrimental changes in meat quality suggest that stress, whether induced hormonally or by exposure to over-heating, caused losses that were as severe as those associated with PSE under field conditions.

(*Key words*: stress; pale, soft, exudative; broiler; meat; nutritional value)

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INTRODUCTION

Meat described as pale, soft, and exudative (PSE) has become a serious quality problem for many poultry processors and consumers. Meat exhibiting PSE has reduced water-holding capacity, protein extractability or denaturation, and textural gel strength properties (Warris and Brown, 1987; Camou and Sebranek, 1991; Northcutt et al., 1994; McKee and Sams, 1997). The degree of myofibril protein denaturation is directly related to water-holding capacity, i.e., as denaturation increases, water content in the meat decreases. When pork or poultry exhibits PSE, it contains myoglobin that has been oxidized to metmyoglobin; thus, such meat exhibits a grayish-pink color with exudative, wet surfaces that reflect more light and have a pale appearance (Warris and Brown, 1987; Barbut, 1993; Ferket and Foegedling, 1994; Fernandez et al., 1994; Northcutt et al., 1994). Pork with PSE has been estimated to lose approximately 5% more water during cooking than normal pork. Thus, the resulting meat is less juicy and tender (Cassens et al., 1975; Fox et al., 1980; Thompson et al., 1987; Santos et al., 1994; McKee and Sams, 1997).

In swine and broilers, PSE is a condition that occurs most often during hot summer months and when there is a continuous increase in muscle metabolism (Froning et al., 1978; Honikel, 1987; Northcutt et al., 1994; Backstrom and Kauffman, 1995; McCurdy et al., 1996; D'Souza et al., 1998). In broilers, skeletal muscle on the breast (pectoralis muscles) and legs (iliotibialis) often exhibit areas that appear to contain damaged fibers. These areas are white, and strands of muscle fibers appearing to have been cooked are readily apparent (Kjolberg et al., 1963; Thompson et al., 1987).

Exposure of animals to adverse environmental conditions evokes adaptive, i.e., acclimative, responses. These responses are generally of two types: specific or nonspecific. A specific response is mounted to alleviate a specific condition. As an example, when body temperature rises, surface blood vessels dilate, which allows greater blood flow to the periphery to ensure dissipation of excess heat (King and Farner, 1962). Specific responses are not stress responses, rather, they are adaptive or acclimative responses.

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Abbreviation Key: ACTH = adrenocorticotropin; C = caloric; CS = corticosterone; CP = carcass protein; CW = carcass weight; M = moisture content; PSE = pale, soft, and exudative; T_a = ambient temperature.

A nonspecific response, regardless of the type of stimulus, acts to increase the animal's overall resistance to stress. Nonspecific responses have been termed physiological stress responses (Selyé, 1956). The most notable is secretion of glucocorticoids by the adrenal cortex (Nagra et al., 1963). The initiation of a chain of events that culminates in secretion of glucocorticoids is recognition of the stimulus at the level of the hypothalamus, which results in secretion of corticotropin-releasing factor (Resko et al., 1964; Salem et al., 1970). Then corticotropin-releasing factor is secreted directly into the anterior pituitary via the hypophyseal portal circulation where it activates the release of adrenocorticotropin (ACTH). After entering general circulation, ACTH is targeted to cells in the adrenal cortical tissue, where it causes synthesis and secretion of specific glucocorticoids. In the case of birds, the major secretion is corticosterone (CS) (Nagra et al., 1963; Frankel, 1970). After CS enters general circulation, it acts on many cells, but particularly muscle cells, to convert proteins to glucose. This process is termed gluconeogenesis and is the major metabolic mechanism by which an animal resists stress and returns to the homeostasis (Siegel, 1995).

Puvadolpirod and Thaxton (2000 a,b,c,d) and Thaxton and Puvadolpirod (2000) recently have described a new stress model in broilers by using continuous ACTH release via osmotic pumps. They documented a wide variety of nonspecific stress responses in broilers and demonstrated conclusively that adrenocortical release of CS evokes gluconeogenesis to provide energy to return to homeostasis.

Poultry processors in Mississippi have observed increased instances of PSE during periods of extreme heat during summer. Two logical questions that have been posed are, "Does heat stress cause PSE in broilers?" And if protein in meat is used to fight heat stress, "Is the loss of meat permanent?" The purpose of this study was to answer these questions. The experimental rationale was to compare classic physiological stress effects, employing the stress model of Puvadolpirod and Thaxton (2000 a,b,c,d) to the more nebulous effects of heat stress.

MATERIALS AND METHODS

Broiler Husbandry

Newly hatched broilers were obtained from a commercial hatchery in Gordo, Alabama. These chicks were reared at the USDA South Central Poultry Research Laboratory on the campus of Mississippi State University. Chicks were raised in metal batteries, and each battery consisted of six cages (dimensions were length = 10 cm, width = 60 cm, and height = 60 cm). Five birds were housed in each cage. All battery cages were located in environmental chambers that were capable of maintaining constant ambient temperature (T_a), RH, light duration and intensity, and air velocity. Feed and water were available ad libitum. Nutritionally complete starter-grower diets were fed throughout the study.

ACTH Treatment

Adrenocorticotropin[4] was administered by continuous perfusion, as described previously (Puvadolpirod and Thaxton, 2000a). On Day 39, all birds in all six batteries had a mini-osmotic pump[5] surgically implanted under the skin overlying the interscapular space. The surgical field was anesthetized locally by perfusion with lidocaine HCl, was then disinfected, and an incision in the skin of approximately 6 cm was made. A pump was inserted under the skin, and the incision was closed with a small wound clamp.

Three cages of birds in each battery received pumps that delivered ACTH. These birds constituted the ACTH treatment. Birds in the other three cages in each of the six batteries received surgical implants of pumps that delivered avian saline (0.9% NaCl). These birds constituted the no ACTH treatment. The ACTH-loaded pumps delivered 8 IU ACTH/kg BW/d for 7 consecutive d. All pumps delivered fluid continuously at 1 μL/h. Blood CS reached a maximum level within 6 h following implantation and remained elevated until Day 8 after implantation (Puvadolpirod and Thaxton, 2000c).

Heat Treatment

From Days 1 through 15, T_a in all six chambers was 30.6 C and RH was 35.5%. On Day 15, T_a in three chambers was reduced to 24 C and 35% RH. These conditions were maintained throughout the remainder of the experiment (Day 55). These birds served as the no heat controls. Environmental conditions in the remaining three chambers were regulated to mimic an average August day in central Mississippi. From 0000 to 0600 h, T_a was 24 C with 35% RH. From 0600 to 1400 h, T_a was increased gradually, such that by 1400 h a maximum T_a of 34 C was attained. From 1400 h to 2400 h, gradual cooling occurred and T_a decreased to a daily low of 24 C. The RH conditions were within 15% of maximum dewpoint temperature at all times. These summertime conditions constituted the heat treatment, and they occurred from Days 15 through 55.

Air velocity at bird locations in the batteries in the no heat and heat treatments was negligible at all times. Likewise, the lighting schedule was 23 h light:1 h darkness in all chambers, and the lighting source was a single fluorescent fixture in each chamber.

Sampling

One bird from each cage of each battery was removed at each time of measurement. Measurements were taken on Days 39, 41, 43, 47, and 55; i.e., on Days 0, 2, 4, 8, and 16 after starts of ACTH and heat treatments. After each chick was removed from a cage, BW was determined, and the bird was killed by cervical dislocation. Head, feet, skin

[4]ACTH 1039, Corticotropin A, Sigma Aldrich Fine Chemicals, St. Louis, MO 63103.

[5]Model 2001, Alza Corp., Mountain View, CA 94039-7210.

plus feathers, intestines, and all other internal organs, with the exceptions of kidneys and gonads, were removed. Because cervical dislocation was the method of killing, the birds were not bled. Each bird was subsequently weighed and this weight was recorded as dry carcass weight (CW). After weighing, the carcass was immediately immersed in an ice bath and chilled for approximately 2 h or until the meat was below 4 C to prevent spoilage. Carcasses were drained and dried on toweling, placed in individual ziplocked plastic bags, and frozen at –20 C for later analyses.

Analytical Procedures

Evaluations of carcasses consisted of visual observation and analyses (AOAC, 1995) for carcass protein (CP) (AOAC 39.1.19), moisture (M) (AOAC 39.1.02), fat (AOAC 39.1.05), ash (AOAC 31.1.01), and muscle caloric (C)[6] contents. The parts were boned and ground, and a composite 25-g sample of tissue was taken with approximately equal allotments from the drumstick, thigh, and breast of each carcass. Values for CP, M, fat, and ash were expressed as percentages, and muscle C content is expressed as kcal/g.

Experimental Design and Statistical Procedures

Four treatments were incorporated in the experimental design of this study. The treatments were designated as follows: 1) no heat plus saline pumps were the controls, 2) ACTH pumps plus no heat were designated ACTH, 3) saline pumps plus heat treatment equaled the heat group, 4) ACTH pumps plus heat were designated as the ACTH+heat group. By selecting one chick at random from each cage of each battery at each time of measurement, the basic experimental unit was a $2 \times 2 \times 3$ factorial arrangement. Main effects were ACTH, heat, and replication. Individual analyses were conducted for each parameter at every period (STATISTIX, 1996). Significant effects attributable to replication were not found nor were significant effects of replication × ACTH, replication × heat, or replication × ACTH × heat. Therefore, data are pooled over replications and reported as a 2×2 factorial design. Statements of significance are based on $P \leq 0.05$. Thus, each mean represents 18 birds.

RESULTS

The effects of ACTH and heat treatment on BW and on CW are presented in Table 1. In this and all other tables, individual treatment means are included at each day of measurement for each parameter. Additionally, pooled ACTH means indicate comparisons of birds treated with ACTH to those that did not receive ACTH, regardless of heat treatment status; whereas pooled heat means likewise are overall comparisons of heat-treated versus no-heat birds, regardless of ACTH treatment status. Body weight was not affected by either treatment, as indicated by pooled heat and pooled ACTH means on Days 0, 2, and 4. However, on Days 8 and 16, ACTH reduced BW. Carcass weight was not affected by either treatment on Day 0, but on Days 2 through 16 ACTH reduced CW, and on Days 8 and 16 heat caused similar reductions in CW.

Total CP and M are presented in Table 2. Total CP was not affected by ACTH on Days 0, 2, and 4, but on Days 8 and 16 ACTH reduced CP. ACTH reduced carcass M at each time of measurement, except Day 0. Heat treatment did not cause reductions in carcass M content at anytime of measurement. These parameters were not affected during or after the stress period.

Muscle C was not affected by either treatment on Days 0 through 4. However, on Days 8 and 16 both ACTH and heat treatment reduced muscle C (Table 3).

ACTH and heat treatment interactions were computed. However, effects attributable to this interaction were not found at any time of measurement for any parameter.

All treatment groups exhibited some degree of stringy muscle, i.e., dehydrated muscle with a pale color. Even though direct measurements were not performed, two conclusions were formed by individuals who are experts in poultry meat processing: the visible appearance of the meat was indicative, but not precisely that of PSE observed at the processing plant, and incidence and severity seemed greater in ACTH and heat treatments as compared to controls. Visual observation of the controls confirmed that there was no apparent damage to the muscle.

DISCUSSION

A more definitive characterization of PSE is mandated if the role of stress in meat quality is to be understood completely. Visible "white" areas of denatured protein in situ, along with a pale and dehydrated appearance of the meat, is the definitive diagnosis of PSE in a poultry processing plant. Carcasses in this study did not have such visible "white" areas, but the meat appeared pale and dehydrated, as compared to meat commonly observed in the processing plant.

Losses in BW and CW by both treatments, as well as reductions in CP, M, and muscle C content by ACTH and CP and muscle C contents during heat treatment, indicate that the treatments indeed caused physiological stress. The metabolic mechanism, which is mounted to provide energy necessary for the homeostatic condition, is gluconeogenesis (Freeman and Manning, 1961; Judge, 1969; Grandin, 1980; Klasing, 1985; Siegel, 1995; Puvadolpirod and Thaxton, 2000a,b,c). Specifically, protein reserves are converted into amino acids and then into glucose, which is readily available to all cells as an energy substrate (Levine and Ursin, 1991). Apparently, turnover of proteins from skeletal muscle sources occurred with both treatments, especially during recovery (Klasing,1985).

In a recent report, Sandercock et al. (2001) demonstrated that acute heat stress caused skeletal myopathy. This condition was evidenced, in part, by increased plasma creatine

[6]Model 12511, isoperibol bomb calorimeter, Parr Instrument Co., Moline, IL 61265.

TABLE 1. Effects of heat treatment and adrenocorticotropin (ACTH) on BW and dry carcass weight in broilers[1]

Age (d)	Treatment	BW (g)			Carcass weight (g)		
		No heat	Heat	Pooled ACTH $\bar{x}$	No heat	Heat	Pooled ACTH $\bar{x}$
0	No ACTH	1,348	1,328	1,338	725	705	715
	ACTH	1,332	1,325	1,328	724	669	696
	Pooled Heat $\bar{x}$	1,340	1,327		724	687	
		SEM range = 26–41			SEM range = 15–21		
2	No ACTH	1,475	1,491	1,483	838	862	850**
	ACTH	1,475	1,510	1,424	802	824	786
	Pooled Heat $\bar{x}$	1,475	1,432		820	816	
		SEM range = 22–48			SEM range = 16–22		
4	No ACTH	1,650	1,584	1,617	922	898	910*
	ACTH	1,544	1,510	1,527	833	824	831
	Pooled Heat $\bar{x}$	1,597	1,547		880	861	
		SEM range = 26–29			SEM range = 24–34		
8	No ACTH	1,961	1,780	1,866**	1,074	981	1,028**
	ACTH	1,735	1,570	1,652	937	850	894
	Pooled Heat $\bar{x}$	1,842**	1,675		1,005*	916	
		SEM range = 28–34			SEM range = 28–39		
16	No ACTH	2,600	2,257	2,428**	1,417	1,321	1,369*
	ACTH	2,010	1,968	2,005	1,146	1,036	1,091
	Pooled Heat $\bar{x}$	2,361**	2,112		1,281*	1,178	
		SEM range = 47–102			SEM range = 79–111		

[1]Treatments were N0-heat = 25 C and 35% RH during the experimental period; Heat = 24 to 34 C and RH within 15% of maximum dewpoint (conditions mimicked hot August day in Mississippi); ACTH = 8 IU ACTH/kg BW/d for 7 d delivered continuously via surgically inserted osmotic pumps; No ACTH = placebo pumps delivering saline.

*, **Pooled treatment mean differed significantly from its accompanying control ($P \leq 0.05$, $P \leq 0.01$, respectively).

TABLE 2. Effects of heat treatment and adrenocorticotropin (ACTH) on muscle protein and moisture in broilers[1]

Age (d)	Treatment	Carcass protein (%)			Carcass moisture (%)		
		No heat	Heat	Pooled ACTH $\bar{x}$	No heat	Heat	Pooled ACTH $\bar{x}$
0	No ACTH	20.1	20.4	20.2	73.0	73.7	73.3
	ACTH	20.5	20.4	20.4	73.6	73.5	73.5
	Pooled Heat $\bar{x}$	20.3	20.4		73.6	73.6	
		SEM range = 0.20–0.40			SEM range = 0.22–0.38		
2	No ACTH	21.2	20.3	20.7	74.2	73.6	73.9*
	ACTH	19.8	20.1	19.9	73.1	72.0	72.8
	Pooled Heat $\bar{x}$	20.5	20.2		73.7	73.0	
		SEM range = 0.35–0.50			SEM range = 0.18–0.58		
4	No ACTH	20.8	19.8	20.3	72.6	72.5	72.5**
	ACTH	20.4	19.9	20.1	71.0	72.1	70.8
	Pooled Heat $\bar{x}$	20.6**	19.8		71.3	72.3	
		SEM = 0.37–0.52			SEM range = 0.41–0.63		
8	No ACTH	20.8	19.8	20.3**	72.6	72.5	72.6**
	ACTH	19.8	19.3	19.5	71.0	72.1	71.6
	Pooled Heat $\bar{x}$	20.3*	19.6		71.8	72.3	
		SEM range = 0.26–0.41			SEM range = 0.52–0.88		
16	No ACTH	21.5	20.0	20.7*	73.6	72.9	73.2**
	ACTH	20.0	19.3	19.5	71.0	72.5	71.7
	Pooled Heat $\bar{x}$	20.6*	19.6		71.3	72.4	
		SEM range = 0.38–0.44			SEM range = 0.44–0.62		

[1]Treatments were No-heat = 25 C and 35% RH during the experimental period; Heat = 24 to 34 C and RH within 15% of maximum dewpoint (conditions mimicked hot August day in Mississippi); ACTH = 8 IU ACTH/kg BW/d for 7 d delivered continuously via surgically inserted osmotic pumps; No ACTH = placebo pumps delivering saline.

*, **Pooled treatment mean differed significantly from its accompanying control ($P \leq 0.05$, $P \leq 0.01$, respectively).

TABLE 3. Effects of heat treatment and adrenocorticotropin (ACTH) on muscle caloric content in broilers[1]

Age (d)	Treatment	No heat	Heat	Pooled ACTH $\bar{x}$
0	No ACTH	5,959	5,976	5,967
	ACTH	5,801	6,096	5,948
	Pooled Heat $\bar{x}$	5,880	6,036	
		SEM range = 61–94		
2	No ACTH	5,776	5,764	5,770
	ACTH	6,206	5,917	6,062
	Pooled Heat $\bar{x}$	5,991	5,841	
		SEM range = 62–78		
4	No ACTH	5,812	5,906	5,859
	ACTH	5,875	5,958	5,917
	Pooled Heat $\bar{x}$	5,844	5,932	
		SEM range = 64–124		
8	No ACTH	6,203	5,710	5,957*
	ACTH	5,657	5,501	5,579
	Pooled Heat $\bar{x}$	5,930*	5,605	
		SEM range = 59–135		
16	No ACTH	6,149	5,610	5,880**
	ACTH	5,506	5,576	5,539
	Pooled Heat $\bar{x}$	5,827**	5,592	
		SEM range = 36–82		

[1]Treatments were No-heat = 25 C and 35% RH during the experimental period; Heat = 24 to 34 C and RH within 15% of maximum dewpoint (conditions mimicked hot August day in Mississippi); ACTH = 8 IU ACTH/kg BW/d for 7 d delivered continuously via surgically inserted osmotic pumps; and No ACTH = placebo pumps delivering saline.

*, **Pooled treatment mean differed significantly from its accompanying control ($P \leq 0.05$, $P \leq 0.01$, respectively).

kinase activity, greater hemorrhage scores, and higher drip losses. This work incorporated several parameters of muscle integrity not investigated in the present study. However, results of both studies indicated clearly that stress is detrimental to poultry muscle quality.

Camou and Sebrenek (1991) and Northcutt et al. (1994) showed that meat exhibiting PSE had reduced water-holding capacity, protein content, and textural strength. Again, these findings do not conflict with findings herein or those of Sandercock et al. (2001), i.e., loss in carcass M and increased drip loss, respectively. Additionally, in the present study the loss in carcass M in ACTH-treated birds occurred even though the meat was ice-chilled for 2 h before samples for analyses were collected.

TABLE 4. Effects of heat treatment and adrenocorticotropin (ACTH) on muscle fat and ash in broilers[1]

		Carcass fat (%)			Carcass ash (%)		
Age (d)	Treatment	No heat	Heat	Pooled ACTH $\bar{x}$	No heat	Heat	Pooled ACTH $\bar{x}$
0	No ACTH	4.08	3.38	3.73	0.96	0.94	0.95
	ACTH	4.19	3.68	3.93	0.93	0.92	0.92
	Pooled Heat $\bar{x}$	4.14	3.53		0.95	0.93	
		SEM range = 0.27–0.42			SEM range = 0.02–0.02		
2	No ACTH	3.76	4.08	3.92	0.91	0.95	0.93
	ACTH	3.54	3.44	3.49	1.02	0.96	0.99
	Pooled Heat $\bar{x}$	3.65	3.76		0.97	0.95	
		SEM range = 0.16–0.41			SEM range = 0.01–0.08		
4	No ACTH	4.91	4.85	4.89	0.96	0.95	0.96
	ACTH	3.92	4.5	4.21	0.97	0.92	0.95
	Pooled Heat $\bar{x}$	4.42	4.68		0.97	0.94	
		SEM range = 0.44–0.47			SEM range = 0.01–0.03		
8	No ACTH	4.57	4.5	4.57	0.95	0.96	0.96
	ACTH	3.9	3.65	3.78	0.95	0.96	0.96
	Pooled Heat $\bar{x}$	4.24	4.07		0.96	0.95	
		SEM range = 0.36–0.44			SEM range = 0.02–0.05		
16	No ACTH	3.4	4.44	3.92	0.91	0.92	0.92
	ACTH	3.4	3.5	3.45	0.95	0.94	0.94
	Pooled Heat $\bar{x}$	3.4	3.97		0.93	0.93	
		SEM range = 0.32–0.45			SEM range = 0.02–0.05		

[1]Treatments were No heat = 25 C and 35% RH during the experimental period; Heat = 24 to 34 C and RH within 15% of maximum dewpoint (conditions mimicked hot August day in Mississippi); ACTH = 8 IU ACTH/kg BW/d for 7 d delivered continuously via surgically inserted osmotic pumps; No-ACTH = placebo pumps delivering saline.

The present findings are indicative, but not conclusive, that stress induced by ACTH and heat treatment cause PSE in broiler meat. Both stressors caused losses in BW and CW and decreased carcass CP and muscle. Additionally, ACTH caused a decrease in M content.

Possibly, longer treatments of ACTH and more acute heat treatments may trigger all overt symptoms of PSE. Nevertheless, stress induced by ACTH and heat caused detrimental effects on yield and protein content of broiler meat without reducing body fat (Table 4). When ACTH and heat treatments were combined, effects were generally additive during the treatment period. Finally, effects on BW, carcass yield, CP, M, and muscle C had not returned to control values after 1 wk of recovery.

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